α-carotene ($p = 0.018$) and β-carotene significantly increased ($p = 0.013$). Older age ($p = 0.025$), higher pre-intervention uncontrolled eating ($p < 0.001$) and plasma carotenoids ($p = 0.009$) predicted weight-loss.
Conclusions: This study provides data which contributes to the characterisation of nutritional biomarkers in obese COPD patients. We have also demonstrated the efficacy of a weight-loss intervention in improving diet quality in this population. Future studies are needed to confirm these findings, and examine the long-term efficacy of the weight-loss intervention.
Funding source(s): John Hunter Hospital Charitable Trust Research Grants Scheme.

EFFECTS OF WEIGHT LOSS AND DIETARY QUALITY ON ENDOTHELIAL FUNCTION – A PILOT STUDY

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Background/Aims: Improved flow mediated dilatation (FMD) has been reported in some but not all weight loss studies. Restricting sodium is known to improve FMD. It is unclear whether weight loss combined with reduced sodium will have a greater benefit on FMD. This study aimed to determine the effects of weight loss and reduced sodium intake on FMD.
Methods: Participants were randomly assigned to two groups, in a parallel design. All participants reduced sodium intake by 50 mmol for one week. Group 1 (weight loss) then used low sodium meal replacements for weight loss, and Group 2 (control, no weight loss) continued their usual diet for 8 weeks. Adherence was assessed using 24 hr urinary sodium excretion. FMD, blood pressure (BP), and 24 hr urinalysis were measured at baseline and at completion of each intervention phase.
Results: Twenty-five participants (14 women; BMI: 34.7 ± 5.9 kg/m^2; age: 42 ± 16 y) were enrolled and 23 participants completed the protocol. FMD (2.3 ± 1.1%, $p = 0.04$, $n = 25$) and DBP (-2.7 ± 10mmHg, $p = 0.01$, $n = 25$) improved after the sodium reduction. During the weight loss phase, mean weight change was -3.0 ± 3.3 kg Group 1 ($n = 13$) and +1.1 ± 1.2 kg Group 2 ($n = 10$), $p = 0.02$. There were no between group differences in FMD during the weight loss phase. Urinary sodium decreased in Group 1 (-87 ± 18 mmol/d, $p < 0.01$) and Group 2 (-95 ± 25 mmol/d, $p < 0.01$) during sodium restriction, and remained lower (Group 1: -67 ± 20 mmol/d, $p = 0.01$; Group 2: -69 ± 24 mmol/d, $p = 0.02$) during the weight loss phase.
Conclusions: These results suggest reducing sodium intake improves FMD but the combination of modest weight loss does not provide additional benefit.
Funding source(s): Heart Foundation and Government of South Australia.

EMU OIL PROMOTES BODYWEIGHT GAIN IN A MOUSE MODEL OF INFLAMMATION-ASSOCIATED COLORECTAL CANCER

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Background/Aims: Patients suffering from the inflammatory disorder ulcerative colitis (UC) have an increased risk of developing colorectal cancer (CRC). Previously, we demonstrated that Emu Oil (EO) reduced inflammation and protected the intestine against UC, NSAID-enteropathy and chemotherapy-induced mucositis. We aimed to determine whether orally-administered EO could reduce the severity of inflammation-associated CRC in mice.
Methods: Mice ($n = 8$ per group) were orally-administered either water or EO (EO1: 80 μL or EO2: 160 μL), thrice weekly. Mice were injected with azoxymethane (AOM), followed by 3 cycles each consisting of 7 days dextran sulphate sodium (DSS) and 14 days drinking water; and culled 3 weeks after the last cycle. Bodyweights, organ data and colonic tumour numbers were recorded. $P < 0.05$ was considered significant.
Results: During the first 2 DSS-weeks, AOM/DSS decreased bodyweight gain compared to normal controls (maximum 23%; $p < 0.05$). However, in AOM/DSS mice, EO2 increased bodyweight gain compared to untreated and EO1-treated mice (maximum 10%; $p < 0.05$) during the 3rd DSS-week until cull. Spleen weight was greater in AOM/DSS-treated mice (water: 0.3 ± 0.03%; EO1: 0.3 ± 0.05%; EO2: 0.3 ± 0.03% relative to bodyweight) compared to normal controls (0.2 ± 0.05%; $p < 0.05$). Thymus weight decreased only in AOM/DSS control mice, compared to normal controls ($p < 0.05$). AOM/DSS resulted in CRC development (water: 10.1 ± 1.7; EO1: 10.3 ± 1.2; EO2: 9.4 ± 1.7 tumour count) compared to normal controls (0 ± 0; $p < 0.05$).
Conclusions: Despite improved bodyweight, emu oil did not decrease the number of colonic tumours. Further studies underway include assessments of tumour size, histological morphometry, apoptosis and proliferation and pro-inflammatory cytokines.
Funding source(s): Cancer Council of Western Australia.

EFFECT OF COMBINED FISH OIL PLUS COENZYME Q_{10} SUPPLEMENTATION ON OMEGA-3 INDEX AND CARDIOVASCULAR RISK MARKERS IN OVERWEIGHT MEN

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Background/Aims: A high Omega-3 Index is associated with a lower risk of sudden cardiac death, and dietary fish oil supplementation has been shown to improve cardiovascular disease (CVD) risk factors. There is little research examining CVD risk factor modification resulting from change in the Omega-3 Index due to fish oil supplementation. We aimed to assess the effects of 12 weeks fish oil supplementation on change in Omega-3 Index together with CVD risk factors.
Methods: Fifty overweight men were randomised to receive fish oil (1728 mg fish oil containing 1000 mg EPA + DHA per day) combined with 200 mg antioxidant (coenzyme Q_{10}), or placebo (2 g olive oil) for 12 weeks. Anthropometry and biochemical outcomes were measured at baseline, six and 12 weeks supplementation. Relationships between change in Omega-3 Index and change in anthropometric and biochemical outcomes were examined by linear regression.
Results: Forty-eight men completed the trial. Baseline Omega-3 Index was high in both the fish oil and placebo groups (7.9 ± 0.4% and 8.1 ± 0.3%, respectively). Fish oil supplementation resulted in a large and significant increase in Omega-3 Index (Δ 2.8 ± 0.3%), whereas placebo supplementation did not (Δ 0.3 ± 0.2%, $p < 0.001$). There was no significant effect of change in Omega-3 Index on CVD risk markers ($p > 0.05$ for all).
Conclusions: In overweight men with a high baseline Omega-3 Index fish, oil supplementation increased the Omega-3 Index but this was not associated with improvement in CVD risk markers.
Funding source(s): Blackmores Australia Ltd.

RESISTIN AND RESISTIN:ADIPONECTIN RATIO PREDICT LUNG FUNCTION IN ASTHMA

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Background/Aims: Adipokines, such as resistin and adiponectin, may contribute to increased asthma risk and severity in obese people. We aimed to examine plasma resistin and resistin:adiponectin ratio in asthmatics compared to healthy controls, according to asthma severity, BMI and gender, following weight loss in obese asthmatics.
Methods: In a cross-sectional observational study of asthmatic adults ($n = 96$) and healthy controls ($n = 46$), plasma resistin and adiponectin were measured. In a separate intervention study, obese asthmatic adults ($n = 27$) completed a 10-week weight-loss intervention and plasma resistin and adiponectin were measured.